MEDICAL AND SURGICAL CONSIDERATIONS IN PYOPERICARDITIS, WITH REPORT OF CASES.[1]

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MEDICAL ASPECTS, BY J. A. SCOTT, M.D.

Pericarditis is usually divided into primary and secondary varieties. Primary pericarditis is so rare that it can be dismissed without further consideration. Secondary pericarditis, or that concurrent with or sequent to other diseases, is that commonly met with in practice. This paper deals only with purulent pericarditis of this type.

Frequency of Pericarditis. This is a difficult matter to decide, for statistics vary considerably. Anders' figures, collected to show the frequency of cardiovascular lesions in croupous pneumonia, show in 275 autopsies the presence of acute pericarditis in 14 cases, or 5 per cent., and chronic pericarditis in 11 cases, or 4 per cent. Riesman, in a study of adhesive pericarditis, analyzed 778 necropsies in the Philadelphia Hospital and found 60 cases of pericarditis, 7.7 per cent.; 20 of these were adhesive; very few were purulent. Breitung, collecting 324 cases of pericarditis at the Berlin Charité, found the following figures: serofibrinous cases, 108; hemorrhagic, 30; purulent, 20 (6.1 per cent.); secondary tuberculous, 24; primary tuberculous, 2; partially adhesive, 111; totally adhesive, 23; ossifying, 2. The proportion of cases of pericarditis occurring in autopsy work is given in von Ziemssen's *Handbook* by various authorities as follows: Duchet, 15.1 per cent.; Chamber, 16.2 per cent.; Willigh, 14.1 per cent.; Taylor, 12.5 per cent. G. E. Still, in an article on "Suppurative Pericarditis in Children," finds in 769 autopsies 28 cases of pericarditis, apparently due to pyogenic infection. Twenty-three of the 28 cases occurred in children under three years of age, and 24 of the 28 showed empyema, thick lymph on the pleura, or acute pleurisy. In 13 cases in which the fluid was examined bacteriologically, 11 were due to pneumococcal infection; of the remaining 15, a large proportion were due to the pneumococcus—empyema, pneumonia, and meningitis being associated.

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Analyzing the mortality statistics of the Pennsylvania Hospital since the opening of the Ayer Clinical Laboratory, I have found 40 cases of pericarditis in 552 autopsies. Ten were obliterative in type; in 13 the exudate was serofibrinous; in 16 (40 per cent.), purulent; in 1 hemorrhagic. The great discrepancy will be observed at once between these statistics and those of Breitung, in which were found but 20 cases of purulent pericarditis in a total of 324 cases of all varieties. Clinically these 40 cases were etiologically as follows: tuberculous, secondary, 7; rheumatic, with acute or chronic endocarditis, 5; aneurysm, cardiac and abdominal, 2; nephritis, general streptococcus septicæmia, cirrhosis of liver, and secondary anæmia, 1 each; and lobar pneumonia, 22. In all these 22 cases pleurisy coexisted with the pericarditis; in 6 cases both the pericarditis and pleurisy were fibrinopurulent; in 4 each process was fibrinous; in 3 cases purulent pericarditis was associated with plastic pleurisy, and in 3 cases the pleurisy was fibrinopurulent, while the pericarditis was fibrinous. In 2 cases plastic pleurisy and adhesive pericarditis coexisted, and in 2 others the pericarditis was purulent, while the pleurisy was serofibrinous. In the remaining case the pericarditis was serofibrinous, and the pleural effusion was bloody.

Again, recognizing the great frequency of purulent pericarditis in croupous pneumonia, I have gone over the total number of autopsies upon that disease and find them to total 76 cases of croupous pneumonia and 5 of bronchopneumonia. In 38 cases of croupous pneumonia, or exactly 50 per cent., pericarditis has been found; in 20 cases, or 52.6 per cent., the exudate was serofibrinous; in 17, or 44.7 per cent., the pericardium contained a purulent exudate; in one case the pericardium was obliterated (chronic adhesive pericarditis). In none of the bronchopneumonias (non-tubercular in origin) was pericarditis present.

ETIOLOGY. Purulent pericarditis is seen in pyæmic diseases—the *streptococcal and staphylococcal infections*—*as a result of* empyema, and in perforation of abscesses in the neighborhood of the pericardium; cancer of the œsophagus, cardiac and aortic aneurysm, etc. It is, I believe, commoner during lobar pneumonia than is recognized, and, as the autopsy records at the Pennsylvania Hospital will show, must be responsible for the death of the patient in not a few cases. The filling of the pericardium with exudate during croupous pneumonia is, I believe, not infrequently overlooked. The diagnosis, however, under such circumstances is a difficult one, as will be detailed later.

BACTERIOLOGY. The variety of micro-organisms found in purulent pericarditis depends upon the primary infection producing the disease. The purulent form is most commonly caused by streptococci, staphylococci, bacilli coli communis, pneumococci, and the tubercle bacilli. There is no reason why the gonococcus and the typhoid bacillus should not play a part in purulent pericarditis.

When a connection is established between the pericardium and the œsophagus or the lung numerous saprophytic micro-organisms may be found.

SYMPTOMATOLOGY. Symptoms are often difficult to interpret correctly, mainly because of the fact that pyopericarditis is commonly only one part of the symptomatology of such serious illnesses as a general pyæmic state, abscesses of the mediastinum, or pneumococcal and other infections.

General Appearance. The patient usually presents an anxious expression and dusky pallor of the skin, with dilatation of the alæ of the nose. Dyspnœa is usually present, at times extreme. The pulse in these cases is quick, with a sharp rise under the finger, rapid and of low tension; very unlike the pulse of an acute infection. At times the large veins of the neck are much distended.

Pain. Common usually over the region of the heart, but of moderate severity; again, it may be intense and agonizing, radiating over the left chest and down the left arm. It is commonly increased by pressure in the epigastric region. During the development of a purulent pericarditis, and while friction rub is present, the pain is apt to be more severe and disappears to some extent with the presence of a large effusion. In many cases pain in the epigastric region is an important symptom; this is accompanied with a sense of constant fulness in the same area; nausea and vomiting are apt to be present in this type. The vomiting is bile-tinged in some cases.

Dyspnœa is usually present and is disproportionate to the amount of pulmonary involvement and the rate of pulse.

Cough. Often dry, frequent, and distressing. Often non-productive. If the pericarditis is associated with pneumonia the sputum may, of course, be blood-tinged. Aphonia and dysphagia may be present.

Temperature. Fever is, of course, complained of to some extent. The temperature is at times but slightly elevated, though the pericardium may be filled with pus. The pulse is weak, small, and may present an intermittency known as the pulsus paradoxus, a condition in which the radial is either greatly weakened or altogether obliterated during inspiration. Delirium in pericarditis of all forms is common. At times it is active, at other times it is low. It is especially to be found in the rheumatic cases with hyperpyrexia.

PHYSICAL SIGNS. Purulent pericarditis at times develops without the ordinary physical signs of the disease being present, or at least discoverable; that is, though carefully sought for, the presence of friction rubs over any portion of the precordial area cannot be detected. In many cases pericardial friction rubs antedate the presence of effusion. This is certainly so in the serofibrinous cases. They are heard most commonly in the second, third, and fourth interspaces to the left of the sternum, perhaps more frequently over the base of the heart than at the apex. They are close to the ear, rough in character; sometimes both coarse and fine sounds can be heard. Heard with the systole and diastole of the heart and not always consistent in length or duration. Pressure increases their intensity. Their inconsistency is their characteristic. Change in position alters the character and time of the sound. At times these rubs can be heard over a large area of the chest; under some circumstances as far as each nipple and down into the epigastric region. Friction sounds, of course, disappear with the presence of effusion into the pericardial sac. Under such circumstances a distinct alteration in physical signs occurs. The apex beat, which heretofore was in its normal position, becomes slightly elevated, perhaps to the fourth space, or may disappear altogether. It may, however, still be palpable in the fourth space. The sounds at the apex are distant and weak. With a normal endocardium no valvular murmurs are heard. Frequently, however, in these cases the causative factor in producing the pericarditis also has caused an endocarditis. Not infrequently the two conditions coexist. Valvular murmurs are therefore to be heard at the areas affected. Coincident with the disappearance of the apex beat occurs an increase in the precordial area of dulness. This is especially noticeable upward to the left of the sternum, perhaps to the second rib and in the fifth and fourth spaces to the right of the sternum. Impaired resonance or dulness discovered in the fifth or fourth interspaces to the right of the sternum, with decided increase of the left border of the cardiac outline and disappearance of the apex beat with weak or feeble heart sounds, is certainly extremely suspicious of fluid in the pericardium. Under such circumstances other physical signs are apt to be present. Owing to the presence of large quantities of fluid in the pericardial sac there is compression of the left lung, especially the lower lobe, in the axillary region. Here at times can be detected a hyperresonant note. The same pressure frequently will produce an impaired or dull note at the extreme base of the left lung. On auscultation at this point the breath sounds may be bronchovesicular or bronchial. This is a pressure consolidation. With physical signs such as these no hesitancy should be felt in inserting a needle into the fourth or fifth space near the left sternum, especially if the absence of the apex beat is demonstrated. In not a few cases fluid will be secured by exploratory puncture in the fourth or fifth right intercostal space. In some cases, however, the physical signs are extremely difficult of interpretation, owing to the presence of complicating conditions. Thus not infrequently in croupous pneumonia there is fluid both in the chest and in the pericardium. Thus in Case II. an empyema of very considerable size dislocated the heart to the left so that the impulse was visible in the fifth interspace one inch outside the midclavicular line. With the evacuation of the empyema the pericardial condition was entirely overlooked, and this probably resulted in the man's demise. In two other cases under observation (Case IV. and one not reported in this paper) there was visible pulsation in the second and third interspaces to the right of the sternum, with the development of (in Case IV.) an enormous shield-like area of dulness between the two nipples, with absolutely clear breath sounds at the bases and axillary region on both sides. These physical signs could only therefore be explained by the supposition that the right side of the heart was adherent to the pericardium or that its pericardial effusion had displaced the heart *in toto* to the right. In the unreported case spoken of, adhesions between the lung and the pleura on the left side were so dense that the presumption is that the suspected pericardial effusion was behind both the lung and the heart, which in turn was displaced considerably to the right.

TEMPERATURE. The range of fever in purulent pericarditis is less than one would anticipate. As previously stated, the presence of pus in the pericardium will at times distinctly modify an already pre-existing high temperature. In the first case studied, while the pericardium was filled with the purulent exudate, the temperature was subnormal for a week. In Cases II. and III. the temperature range with the pus in the pericardium was between 98° and 100° F., and 100° and 102° F., respectively. In Case IV. the temperature was high. It is difficult to state the exact time of the onset of the pericarditis.

PULSE. The pulse is weak and of low tension from the start, irregular at times; in some cases influenced by respiration, either partially or totally obliterated. The paradoxical pulse was present in Case I.

BLOOD. A leukocytosis is produced in all cases. If, however, the pus has been present for any length of time in the pericardium, the leukocytes have a tendency to fall again toward, but do not reach, the normal. In cases with pre-existing leukocytosis—and this is found in practically all patients with purulent pericarditis—a hyperleukocytosis is added. In Case I. the leukocytes jumped from 6600 on the tenth day to 21,000 on the sixteenth day, and fell slowly to 17,050 on the twenty-eighth day. Three blood cultures were negative. In Case II. a leukocytosis of 22,700 was present on the fourteenth day. The pericardium at that time was probably full. On the twentieth day with both pericarditis and empyema present the leukocytes were 12,000. The blood was not counted after operation; no blood cultures were taken. In Case III. the leukocytes on the sixth day were 16,400; on the tenth day, 30,800. At this time pericarditis was diagnosed. Blood cultures on the third and fifth day were negative, but a positive culture was found on the tenth day after pericarditis was present. In Case IV. no cultures or counts were made. The urine is much reduced in quantity in all cases; usually contains albumin, no sugar, and on microscopic examination shows rather numerous casts; in our case granular casts have predominated, though hyaline were also found. The urea output in Case II. (the fatal case) was 30 grm. in the twenty-four hours.

In conclusion, in pneumonia the cardiac outline should be carefully mapped out at the onset. Any changes either in the size or position of the heart should carefully be watched for. With the development of an empyema more rigid scrutiny of the heart should be insisted upon. The presence of dulness in the fifth right interspace or the dislocation of the heart either to the right or to the left side without a coexisting empyema, should lead one to suspect the presence of fluid in the pericardium, and careful selection of the spot for exploratory puncture should be made.

DIAGNOSIS. Pyopericarditis is to be diagnosed by the following signs: The presence of a primary infection capable of producing pus; the development of a rapid, weak, and at times intermittent pulse, cough, and dyspnœa; pain over the heart and epigastric region of severe nature; a temperature of mild degree, and mental symptoms of varying intensity. Associated with these symptoms pericardial friction rubs over the base or the entire cardiac area may be heard. They are at times altogether absent (Cases I. and II.); then follows the rapidly increasing, pear-shaped precordial area of dulness, extending upward to the left second rib, to the right in the fifth, fourth, and third intercostal spaces, at times reaching the right midclavicular line, and to the left to and beyond the left midclavicular line. It is important to note that the apex beat, if present, is within this area of dulness and not at its extreme limits. The shape of this dull area varies with the amount of fluid in the sac. If the quantity is not large the dull area is pear-shaped with its base exaggerated. If the quantity is large the dulness is almost circular or ovoid at its upper border, the centre of the bow being the sternum. The lower dulness merges with that of the liver or even the spleen, but more often with the dulness or flatness of the consolidated lung or that produced by fluid in the pleura. The apex is either lifted to the fourth space or disappears altogether, or the entire heart is bodily displaced to the right or left, according to circumstances.

The sounds at the apex grow distant and faint and at times can be heard with difficulty. The left axilla presents a hyperresonant note with exaggerated breathing; the left base may show a spot of compression dulness with bronchial breathing. To clinch the diagnosis an exploratory puncture should be made in that portion of the dull area farthest away from the ascertained position of the heart, preferably in the fourth or fifth space to the left of the sternum.

DIFFERENTIAL DIAGNOSIS. 1. Serofibrinous pericarditis. 2. Acute dilatation of the heart. 3. Acute catarrhal gastritis. 4. Right-sided or left-sided pleural effusion or empyema, either free or loculated. Some unusual conditions have been mistaken for pericarditis, such as mediastinal tumor, acute endocarditis, cases of aortic regurgitation, and we have recently heard that perforation of the bowel was diagnosed by two very competent observers, the man's condition, however, preventing operation, and the autopsy showing the presence of a purulent pericarditis.

Before considering any of these points in diagnosis the following anatomical belief usually stated in text-books is questioned: That the heart usually or always, in the presence of large quantities of fluid in the pericardium, is pushed away from the chest wall, with the consequent disappearance of the apex beat and weakness of sounds. The heart is fixed at its base by the attachment of its vessels, and this leaves the apex and the ventricles as the movable portion to be effected by pressure from all sides or any side; if, however, adhesions already exist between the pericardium and the heart on either the right or left side a lateral dislocation of the heart will occur; or pressure exerted upon the filled pericardium as a whole may cause a lateral dislocation of the heart; perhaps other unknown conditions within the pericardium may produce the same lateral displacement. We hope in the future to be able, with the kind assistance of Dr. Longcope, to experimentally verify some of these views; as yet we have not been able to do so.

1. Serofibrinous pericarditis is usually the result of rheumatic infection or concurrent with tuberculosis or Bright's disease. The history is of decided importance as the causative factor. The symptoms and signs are, of course, much the same as in purulent pericarditis. You have not, however, the usual chest complications seen in the purulent variety. The temperature is apt to be but slight, and the leukocytosis does not reach such a grade as in the purulent form. This fact, however, is not to be relied upon. Cardiac dilatation is common in the rheumatic pericarditis and endocarditis frequent. Dilatation of the heart is not common in purulent pericarditis. The physical signs in both conditions are similar except that we must recall the fact that purulent pericarditis is seldom present without a purulent pleurisy, a pneumonia, or meningitis, or pus or ulceration near the pericardium. Exploratory puncture settles the doubt at once.

2. Acute dilatation of the heart is a common feature of chronic valvular disease and rheumatic pericarditis, but according to Still is uncommon in purulent pericarditis. It is attended with weak, rapid, and irregular pulse. The cardiac impulse, however, is visible, wavy, and occupies several interspaces, although at times it is not palpable; the impulse may be irregular, now weak and now strong, and can be perceived in the epigastric region. The heart sounds are heard, the first short, sharp, but weak, and not either distant or absent. There is a decided increase in the area of cardiac dulness, which, however, does not extend so high as in pyopericarditis. The second sound is sometimes split with so-called "gallop rhythm."

Careful mapping out of the heart dulness shows the mobility of the heart. In dilatation there is a tendency to venous congestion and dropsy; the liver increases in size, the kidneys fail to secrete, and the lungs fill up. The *x*-ray examination is a decided help, showing in dilatation a fixed quadrangular shadow; in pyopericarditis a movable upper line of dulness. The blood count will show the absence of leukocytosis in dilatation. When dilatation of the heart occurs in rheumatic pericarditis in a case in which friction rubs have been easily demonstrable, the diagnosis becomes extremely difficult. It rests mainly on the points mentioned above, yet we have seen the diagnosis of acute pericardial effusion with request for immediate opening of the pericardium made by a most able diagnostician, and the operation show an acute cardiac dilatation.

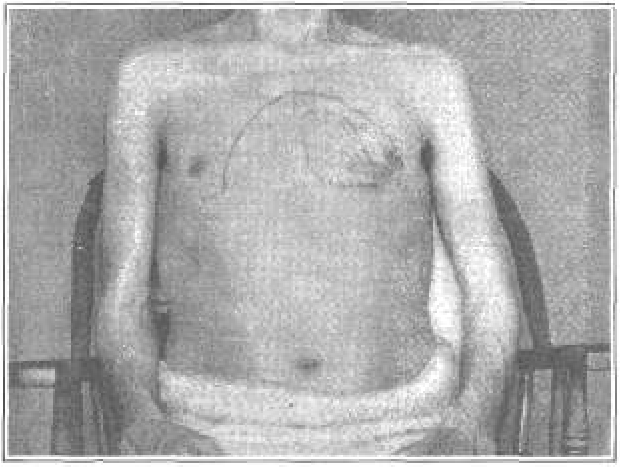
3. Acute catarrhal gastritis presents the history of some causative factor, such as the ingestion of indigestible food, drink, or irritating substance. The vomiting is more or less constant and the pain confined to the gastric area. The temperature is usually high and cough and dyspnœa are not present. The cardiac outline is normal in size and the heart sounds well heard. The blood is of little help, as a leukocytosis may be present in acute gastritis.

4. Pleural effusion, either free or loculated. It is taken for granted that the reader is familiar with the signs of free fluid, be it either serum or pus, in the chest. When this is present in sufficient quantity so that the lower limit of dulness reaches the anterolateral portions of the chest and impinges upon the normal heart dulness, we have a condition which presents many difficulties in diagnosis. If the case has been seen from the start and the size of the heart and the character of its sounds carefully noted, the diagnosis is perhaps not difficult, but when brought face to face for the first time with the presence of large quantities of fluid in the chest with the dislocation of the heart either to the right or to the left and apparent increase in the outlines of the pericardial sac, the diagnosis becomes extremely difficult. Under such circumstances the liberation by either aspiration of the serum or resection of a rib and thorough drainage of the pus is, we think, essential to an opinion. With the relief of the fluid the heart will return toward its normal site. If there remain an unusual area of precordial dulness there is a great probability that the pericardium is also filled. The great frequency of involvement of both the pleura and pericardium in pneumonic cases must be remembered. Of course, change in position of the patient will in large pleural effusion or empyema alter the upper line of dulness. This will, however, give you but little information with regard to the pericardium. Loculated empyema is usually latent and extremely difficult to localize. Unless large it is not apt to dislocate the heart except when present in the anterolateral part of the chest. When so situated the heart is dislocated to the right or left and a visible impulse and apex beat can still be

seen. The symptoms of a loculated empyema, cough, fever, and sweats, with the consequent leukocytosis persist. The pulse remains good, the heart sounds are readily heard and are not weak. There is no evidence of pericardial friction.

CASE I. (Reported in *New York and Philadelphia Medical Journal*, January 30, 1904.)—An Italian laborer admitted to hospital on the sixth day of pneumonia of the right base. Consolidation of left base on ninth day, with an extensive pleurisy. Pericarditis diagnosed on the thirteenth day. Paracentesis performed with immediate relief. Reaccumulation and formal operation on January 31st under local anæsthesia. Slow but complete convalescence at the end of six weeks' time.

M. D., an Italian laborer, aged thirty-six years (Hospital No. 3215), admitted to the Pennsylvania Hospital on January 4, 1903, with history of six days' illness, beginning with a chill, cough, and pain in the right side. On admission physical signs of consolidation of the right base were present. From the start the pulse was weak and heart sounds feeble, though the heart was not displaced and cardiac outline normal. Respirations were rapid (48 to 56), and the leukocytes (ninth day) 8800. On the ninth day the base of the left lung showed signs of consolidation with extensive pleurisy, which was especially marked in the left mammary region. The temperature for the first four days ranged between 103° and 102° F., and gradually fell by the twelfth day to 100° F. in the morning with an evening rise of a degree or more. This low temperature was maintained from this time on. The right base cleared slowly. By the thirteenth day the patient appeared very sick, with grayish



CASE I.—The upper line shows the outline of dulness when the pericardium was filled with purulent fluid ; the smaller outline or cardiac dulness six weeks after operation.

pallor, dyspnœa, and weak, rapid pulse. No pericardial friction was heard, but the pericardium was considered involved by the thirteenth day, empyema of the right base having been excluded by puncture. The apex beat now was neither visible nor palpable and there was impaired resonance in the fifth and fourth interspaces to the right of the sternum. This area of cardiac dulness increased, especially on the right side. The cardiac sounds were very indistinct. The epigastric region was full and very tender. The paradoxical pulse was marked on the eighteenth day of illness, January 16th. The leukocytes, before not increased, had now risen to 18,600. An exploring needle inserted in the fourth right intercostal space showed the presence of light-greenish, turbid fluid, which on culture showed a pure pneumococcic growth. The pericardium was aspirated at the same spot the following day and 420 c.c. of seropurulent fluid were withdrawn, with great relief to the more urgent symptoms. In a week's time rapid reaccumulation occurred, and, as was expected, exploratory puncture now showed thick, yellowish pus in the pericardium. The patient refused operation primarily. Permission to drain the pericardium having finally been secured, Dr. Le Conte operated on January 31st. Chloroform anæsthesia was at first attempted, but resulted in such struggling and cyanosis that local anæsthesia with cocaine was at once substituted and the fifth space incised at the site of the normal apex beat. The pericardium was readily opened and between a pint and a quart of non-odorous pus slowly escaped. The laboratory examination of the fluid showed many leukocytes and pneumococci.

The man was very ill for two weeks after operation, with weak, irregular pulse and but slight fever. From February 22d to 27th there was slight œdema of the face, back, and ankles present, though the urine during this time was free from casts, only leukocytes being present. At no time subsequent to the operation were pericardial friction sounds heard. The drainage tube was removed in twenty-six days and the patient was permitted to leave the hospital on February 23d, fifty-one days after operation.

It is interesting to note in this case the very mild degree of fever present while the pericardium was filled with seropurulent and purulent material.

This patient was seen December 31, 1903, almost a year after operation. He looks well and has gained flesh, and has been doing light work in a store. He states that he is sleepy a good deal of the time and has, on deep inspiration, some pain over the heart and in the back. No dyspnœa or cough is present. Appetite and digestion are good. Pulse is 76, small in volume, and very compressible. There is no œdema of feet. Liver is normal in size.

Inspection of Chest. No venous phenomena of neck; no retraction of interspaces over the cardiac area or behind (Broadbent's sign). The outline of the cardiac dulness is normal. The apex in the erect position is in the fifth interspace within the midclavicular line. It is definitely movable with change in position. When he lies upon the right side the apex falls at least an inch toward the sternum, and lying on the left side pulsation is visible and palpable one inch outside of the nipple.

Auscultation. First sound is rather short, somewhat liquid, and with weak muscular tone. Second sound is clear; pulmonic sounds are distinct and normal, while the aortic sounds are rather weak and clear.

The features of interest in this case are:

1. The absence of leukocytosis during the pneumonia and its development with the pericarditis.
2. The absence of the pericardial friction sounds, both before and after operation.
3. The absence of fever, subnormal temperature with pus in pericardium.
4. Recovery without the physical signs of an adhesive pericarditis.

CASE II.—This case is one of lobar pneumonia complicated with empyema resulting in operation, with death eight days after operation; pyopericarditis overlooked.

The man, S. J. B., aged fifty-two years, was admitted to the Pennsylvania Hospital on December 21, 1903 (No. 2860). His family history is unimportant. Except for a pleurisy four years ago, which lasted three months, has had no serious illness. No venereal history. Formerly drank excessively, but periodically. Smokes freely. Is an attendant for feeble-minded children in Vineland.

Eight days before admission to the hospital he had a severe chill, which was followed by pain in the right side. He had a slight cough with little whitish expectoration. On admission he presented unusually small but equal pupils, urinous breath, with dry, parched tongue. Both the apex and base of the right lung are consolidated; the middle lobe escapes. The liver is increased in size. The abdomen is large; distention marked. The heart is either dislocated or increased in size, the right border being the right edge of the sternum, the left border anterior axillary line, the upper border the second interspace. The sounds are heard distinctly, but are not strong. The apex beat is palpable two inches outside the midclavicular line in the fifth space. No murmurs are present. The temperature on admission and for the first forty-eight hours was about 102° F. On the ninth day there was a fall to 100° F., with subsequent rise (pseudocrisis), and on the eleventh day to 99° F. From this time the temperature is but slightly elevated, about 100° F. The most extreme tympanites developed, which, it is interesting to note, was relieved by eserine. By the twentieth day of the sickness, or twelve days after admission, the signs at the right base indicated fluid. At this time, however, the leukocytes had fallen from 22,700 on the fourteenth day to 12,600. Exploratory puncture on February

5th showed the presence of pus. At this time it was noted that there was impaired resonance in the third and fourth interspaces to the right of the sternum. It was, however, supposed to be due to the line of fluid. The fifth rib in midaxillary line was resected by Dr. Le Conte and showed the presence of a large quantity of thick, yellowish pus in the right pleural sac. He did not do well after the operation, the pulse growing weaker and dyspnœa increasing; the temperature remained low. I regret to say that I did not see him after he was operated upon.

The autopsy showed, in addition to an unresolved pneumonia of the upper and portions of the middle and lower lobe, that the right pleural cavity was well drained, but that a purulent exudate filling the pericardium had been entirely overlooked. The pericardium contained 600 c.c. of thick, yellowish pus.

CASE III.—Croupous pneumonia of the left base with the development of pericardial friction on the sixth day of illness; partial disappearance of the friction, with disappearance of the apex beat and great increase in the area of cardiac dulness. No evidence of fluid at the base at this time; incision into the pericardium, evacuation of pus; second operation in eleven days after the first, with drainage of some pus from the left pleura; rapid convalescence and recovery.

D. S. (No. 3141), aged thirty-three years, an Italian laborer, admitted to the Pennsylvania Hospital on January 14, 1904, complaining of pain in the left chest and back, with cough and dyspnœa, and fever of twenty-four hours' duration. On the second day it was evident he had consolidation of the lung involving the left base, faint bronchial breathing being heard at the extreme base and in the anterior axillary line. From the start the pulse was rapid, weak, and compressible. The man was very ill with active delirium. Complained much of pain, especially in the epigastric region and around the left costal margin. On admission the heart dulness was normal in size, no friction sounds were heard, and the heart sounds at the apex were very weak. On January 18th, or the sixth day of the illness, pericardial friction sounds were heard in the left third, fourth, and fifth intercostal spaces. The apex was not visible but palpable in the fourth interspace. By this time bronchial breathing was readily heard all over the left base. On the ninth and tenth days friction sounds were heard at the apex and to the right of the sternum; by the twelfth day there was dulness in the interspaces to the right of the sternum, with very weak pulse, dulness, and absent tactile fremitus in the left axilla. There was no evidence of fluid posteriorly. The leukocytes on the third day of the illness were 17,900; on the seventh day, 16,400; on the tenth day, or the period during which pericardial friction and increase in the area of cardiac dulness occurred, 28,000.

The temperature ran up to 103° F. until the seventh day, when a pseudocrisis occurred with a fall to 100° F., subsequent rise and fall, and on the ninth day to 99° F. The pericardium was considered filled on the twelfth day. An exploring needle was introduced in the fifth interspace one inch within midclavicular line, and purulent fluid readily secured. Operation was performed within one-half hour under Schleich solution by Dr. Le Conte.

About a quart of pus welled through the incision in the fifth interspace. Upon sudden paroxysmal coughing a large quantity of pus, estimated two quarts, was suddenly expelled from the chest through the incision. It seemed as though an empyema must have ruptured through the incision. It is estimated that three quarts of pus were removed, which showed on culture pure pneumococcus growth. For eleven days the temperature remained between 100° F. in the morning and 102° F. in the evening, with rather rapid pulse (112 to 116); respirations about 32.

Unfortunately, the wound was permitted to close up about six or seven days after this operation. The case about this time was transferred to Dr. Harte's care, and after consultation with him it was considered advisable to reopen the chest to establish drainage, this time into the pleura, with the result that a small quantity of pus was recovered. This was done on February 6th and the twenty-fifth day of the illness. From this time on his convalescence was rapid and complete.

This case has been the subject of considerable discussion among his physicians; it has been questioned if the primary operation opened the pericardial sac, and the opinion expressed by those who did not see the case medically was that we are here dealing with an empyema and not pyopericarditis; that is, a loculated empyema was present with dislocation of the heart to the right side, and that the operation opened up the pleural sac and not the pericardium.

CASE IV. (Seen in consultation with Dr. Richard H. Harte in the surgical wards of the Pennsylvania Hospital.)—N. M., an Italian laborer, aged twenty-two years. Both of his feet were frozen and he subsequently developed moist gangrene. Operation (amputation of both feet at lower third leg) after twenty-four days. Profoundly septic at this time. Sloughing of flaps, with granulation. Tardy convalescence. From the middle of February and through March the development of temperature with extensive increase in the precordial area of dulness; lungs clear. Operation, with drainage of the pericardium, on March 14th, with gradual convalescence.

N. M., aged twenty-two years, laborer, admitted to the Pennsylvania Hospital on December 21, 1903. During the cold weather of December this man slept in a barn, in which he was found on the day of admission with both feet frozen. On admission they were perfectly cold and of slate color to about four inches above the ankle. In twenty-four hours blebs formed over both heels, with return of normal color to the skin above the ankles. Moist gangrene developed in several days' time. By January 10th the lines of demarcation present over both insteps with the development of septic temperature. He refused operation, but finally double amputation was consented to and done on January 14th. The flaps, however, did not do well, sloughed and healed slowly by granulation. By January 29th the temperature reached and remained normal for some time. During February, however, there was a gradual increase in temperature without any unfavorable signs. Pulse and respirations increased in frequency with sweats at intervals. No pain or cough or chill. It is noted, however, that the heart is dislocated to the right and visible pulsation is present in the third, fourth, and fifth interspaces, and the apex of the heart seems to be almost at the right nipple. On March 3d, examination shows that the cardiac dulness extends one inch to the right of sternum. The heart's action is embryonal. There is no cough or expectoration. On March 13th Dr. Harte asked me to see this case with him. There was present a large circular area of dulness over the præcordia, extending one and a half to two inches to the right of the sternum, to the second rib and outside the left midclavicular line. No visible pulsation is present. The heart sounds are loudest to the right of sternum. No friction is present. Paradoxical pulse not present. No venous phenomena in neck. The base and axillary region of left lung are clear. A needle inserted into the fifth interspace within the midclavicular line secures light-yellow, purulent fluid, which on examination shows the presence of streptococci. The following day Dr. Harte resected the fourth and fifth ribs within the nipple and secured a large quantity of pus. The man has slowly convalesced, but the heart has not returned to its normal position. There is still visible pulsation in the third right space, while the apex seems to be in fourth space (adherent pericardium?). No Broadbent sign. The points of interest in this case are:

1. The hæmatogenous or embolic origin of the pericarditis.
2. The unusual delimitation of the pericardial outline.
3. The absence of cough and pain.
4. The differential diagnosis between purulent pericarditis and dislocation of the heart by loculated empyema.

SURGICAL CONSIDERATIONS, BY ROBERT G. LE CONTE, M.D.

In presenting the surgical side of pyothorax the limited time at our disposal will prevent a discussion of the subject in full, and we therefore propose to deal only with three questions of importance to the surgeon: 1st, exploratory puncture for diagnostic purposes; 2d, anæsthesia; 3d, intercostal incision or excision of costal cartilages.

1. *Exploratory Puncture.* Purulent pericarditis cannot be definitely diagnosed unless the fluid is in hand for inspection. For this

reason exploratory puncture is an essential procedure in making the diagnosis. As we cannot tell the presence of pus by the degree of severity of the symptoms, the feeble pulse, and cardiac action, etc., therefore a formal operation for opening the pericardium should not be undertaken until pus has been proved present. One drop is sufficient for a diagnosis, and this can usually be obtained with a slender needle similar to the ordinary hypodermic needle, but somewhat longer.

The three principal dangers in exploratory puncture are wounding the heart, opening a large bloodvessel, and infecting the pleura if septic material be encountered in the pericardium.

A wound of the heart is grave according to its size and depth, but a small wound need not of necessity cause any symptoms. The cavity of the ventricles has been aspirated and life saved in acute dilatation of the heart, and it is a recognized and legitimate surgical procedure. If, therefore, a small needle is used no serious complication need result if the heart is punctured. When the heart is adherent to the anterior surface of the pericardium, or when the diagnosis of pericardial distention is at fault, there is great probability that the heart muscle will be penetrated by the needle. Should the needle puncture the heart it would readily be recognized by the motion imparted to it.

For the same reason the dangers resulting from the wounding of a large bloodvessel are insignificant, and need not be regarded if our needle is small. We can readily avoid the usual position of the internal mammary vessels, and no other vessels need be feared except in the presence of some anomalous distribution.

To appreciate properly the dangers of invading the pleural cavity during paracentesis we must turn for a moment to the anatomy of the part. The anterior mediastinal space, equivalent to that portion of the pericardium which is not covered by the pleuræ, practically commences at the level of the upper border of the fourth costal cartilage and extends downward to the diaphragm. The left pleura recedes much farther from the median line than the right, so that usually the edges of the left pleura do not reach the border of the sternum. For the purpose of obtaining some definite knowledge on the width of the anterior mediastinal space, 11 observations were made on the post-mortem table, and we are indebted to Dr. W. T. Longcope, the pathologist of the Pennsylvania Hospital, for the careful measurements and notes of these cases. From these observations we judge that the average width of the anterior mediastinal space, on a level with the fifth costal cartilage, is from 4 to 5 cm.; but that the space is quite variable is shown by the measurements, in one case being 8 cm. and in another 1 cm. In the latter case, with a width of 1 cm., the edge of the left pleura just reached the border of the sternum. In two other cases, where the widths were 3 and 4 cm., respectively, the left pleura reached nearly to the edge of the sternum. In the remaining cases the border of the pleura was some distance from the sternum. The right pleura in every instance reached beneath the sternum.

In this region the left pleura is rather firmly attached to the triangularis sterni muscle, but can easily be dissected from it. Its attachment to the pericardium is a little more firm, but in a normal subject it can readily be separated until it dips over the side of the pericardial sac. For some distance, then, dependent upon the position assumed by the pericardium in the left chest, these two layers of parietal pleura are in close apposition. The substance of the membrane is so thin, frail, and translucent that it would be hard to conceive of any inflammation which involved the pericardium that would not also extend to both layers of the overlying pleura and obliterate the pleural space in this region.

The pericardium, although it does not contain elastic fibres, is capable of very great distention, and its cavity will hold quite two litres (Osler). The lungs are displaced and may be thrust far backward and outward in the presence of such distention, but the pleuræ, on account of their attachments, keep their usual relations to the anterior thoracic wall. That pathological lesions change the pleural relations of the pericardium very little is shown by the dissections of Delorme and Mignon (*Revue de chirurg.*, December, 1895, p. 830) in cases of pericarditis and in bodies where fluid was injected into the pericardium. We cannot compare distention of the bladder and its concomitant displacement of the parietal peritoneum with distention of the pericardium, for the peritoneum in the region of the bladder is very loosely attached to the anterior abdominal wall, while the pleural attachment is much more firm to the triangularis sterni muscle and still more firm to the pericardium. We therefore agree with Roberts in believing that Fowler is in error when he states that the boundaries of the pleuræ are greatly displaced in pericardial distention. As the pericardium probably distends equally in all directions the anterior mediastinal space must be broadened to some degree by this portion of the pericardium distending proportionately with the remainder of the walls of the sac. The more the pericardium is distended the greater will be the surface of pleural contact, and when this surface adheres the larger will be the operative field where the pleura can be crossed with safety.

To return to the observations on the cadaver, pins were thrust in the fourth and fifth interspaces to the right and left of the sternum, hugging the border of the sternum as closely as possible. It was found that on the left side the pins traversed the cavity of the pleura in both the fourth and fifth interspaces in one subject, where the breadth of the anterior mediastinal space was 1 cm.; that in two other subjects, in which the anterior mediastinal space was respectively 3 and 4 cm. broad, the pins in the fourth and fifth left interspaces grazed the pleura, but did not penetrate its cavity. In the other eight cases the pins entered the pericardium some distance from the pleural border. On the right side, in every instance, the pins passed through the pleura before reaching the pericardium.

With such anatomical data we conclude that the point of election for paracentesis, or exploratory puncture, is in the fourth or fifth left interspace, as close to the edge of the sternum as the needle can be made to enter. In this position there is the smallest chance of penetrating the pleura, and the internal mammary vessels should lie well to the outer side of the needle. If exploratory puncture is practised in the fifth right interspace, the point suggested by Rotch, the pleura will almost invariably be traversed. Puncture in the space to the left of the xiphoid cartilage necessitates passing through the diaphragm, and therefore dangerously close to the peritoneum.

The ordinary hypodermic syringe, armed with a needle an inch and a quarter or an inch and a half long, is introduced in the fifth left interspace. If no fluid is withdrawn, and there is no evidence of penetration of the heart by motion imparted to the needle, the fourth interspace should be tried. If this also fails to demonstrate fluid, the same interspace one and one-half or two inches from the left border of the sternum should be tried. If septic material is not found, there can be no danger of infecting the pleura, even though its cavity is invaded. If septic material is present in the pericardium the overlying pleural space is almost certain to be obliterated, and under such circumstances the needle can pass through it without danger of further infection.

2. *Anæsthesia.* In the presence of a large collection of fluid, with feeble heart sounds, the administration of a general anæsthetic becomes very dangerous. In Case I. the patient after four or five inspirations of chloroform became very cyanosed, with feeble, fluttering pulse, and death seemed imminent. Under such circumstances we are forced to use a local anæsthetic, and our experience with infiltrations of Schleich's fluid has proved most satisfactory. In such cases where the heart action has not been seriously interfered with, ether would seem to be the best for general anæsthesia.

3. *Intercostal Incision and Excision of Costal Cartilages.* The choice of operation will be very greatly influenced by the condition of the patient. If a general anæsthetic is contraindicated, excision of a costal cartilage will be dangerous, for under local anæsthesia it cannot be done without causing considerable pain, and severe pain would be very depressing to the heart. Under such circumstances our choice will be limited to an incision through an intercostal space, or aspiration of a large part of the fluid for the purpose of temporarily relieving the embarrassed heart and permitting later the giving of a general anæsthetic.

Incision is certainly as safe as aspiration with a large needle or trocar, and it frequently permits of a recovery without further operative procedure, while aspiration necessitates a later operation.

If the pericardium is much distended with pus the overlying pleural cavity is almost certain to be obliterated, and incision through it can be made without risk.

The point of election for incision is the fourth left interspace, beginning one inch from the sternal border and extending to the normal position of the apex beat of the heart, about one inch internal to the anterior mammary line. The tissues are infiltrated with Schleich's fluid and the incision gradually deepened. When the pleura is adherent it cannot be recognized; but if its cavity is free, and the incision has been carefully deepened, air will rush in as soon as it is penetrated, and before the pericardium has been opened. When pus is reached it is allowed to flow out slowly for fear of embarrassing the heart by the sudden relief of pressure. A rubber drainage tube is inserted and an ample dressing of gauze applied. This is sufficient for the time being, and the question of flushing the pericardium or resecting the costal cartilages may be put off until the physical signs show that the drainage is not good or that the tube is plugged with membranous lymph.

Intercostal incision produces no shock; it is the simplest procedure in grave cases; it permits of a more extensive operation at a later period if found inefficient; it permits the recognition of non-adherent pleural surfaces before the pericardium is opened, and in the majority of cases it will lead to a cure where a recovery is possible from an operative procedure. The question of the recovery of the patient is more dependent upon the cause of the pyothorax and the pathological lesions present in other portions of the body than upon the choice of an operative procedure.

The deductions drawn by Reichard ("Zur Kasuistik der Operationen bei Pericarditis," *Mitteilungen aus den Grenzgebeiten der Medizin und Chirurgie,* No. 7, p. 356), in advocating resection of a rib instead of incision in the intercostal space, do not seem to us to be justified. He collected 33 cases, in 19 of which incision was practised and in 14 resection of a costal cartilage. Of the 19 cases 6 recovered and 13 died, and of the 14 resections 8 recovered and 6 died. From this statement of figures he concludes that the safest procedure is resection. If, however, we examine these cases more carefully we will see that of the 13 cases dying after incision 1 died on the table, 2 others within twenty-four hours, a fourth while the pericardial cavity was being flushed after operation, a fifth some weeks later, following a double empyema operation, and a sixth from joint empyema. These 6 cases would probably just as surely have died after resection, and one is led to believe that perhaps their condition was so desperate that so formal an operation as resection was not used because too prolonged and dangerous. If, then, these 6 failures are subtracted from the 13 deaths, we would have 6 recoveries and 7 deaths following incision. Also, when we examine the cases that recovered under excision, we find that in 1 the disease was primary in the pericardium, and that 2 others were the result of stab wounds—in a sense, therefore, primary also. Such favorable cases would probably have recovered as rapidly with simple incision, and, if so, by removing them from the tables of the recoveries we would have 5 recoveries with 6 deaths. In other words, the statistics of the two procedures may be regarded as very nearly equal in the same class of cases.

If excision becomes necessary we are inclined to think that the chondroplastic flap suggested by Roberts is better than the removal of one or two costal cartilages. Roberts cuts his flap through the fourth and fifth costal cartilages and turns it upward, using the soft tissues of the third interspace as a hinge. On the normal cadaver it gives a good exposure of the internal mammary vessels and readily permits of their ligation if they cross the field of operation, and the border of the left pleura is easily recognized and can readily be dissected free and pushed aside if its surfaces are not adherent.

CONCLUSIONS. 1. Purulent pericarditis is quite frequently overlooked in lobar pneumonia and in other pyæmic states.

2. Its presence modifies the pre-existing disease to a considerable extent.

3. In diseases with high temperature the presence of purulent pericarditis depresses the fever range and increases the respiratory and pulse rate without sufficient clinical evidence of trouble elsewhere than in the pericardium.

4. It is yet to be proved that the heart, in case effusion is present in the pericardium, is always dislocated backward and upward. It is probable that in some cases the presence of fluid in the pericardium dislocates the heart either to the right or to the left. (This, of course, will occur if previous adhesion between the heart and the pericardium has occurred on either the right or the left side.)

5. The diagnosis once made, the only treatment should be incision and thorough drainage. (Paracentesis will at times relieve temporary urgent symptoms and should be performed without the slightest fear.)

6. That exploratory puncture can be made with safety, and is essential for diagnostic purpose before operation is undertaken.

7. That such puncture should be made in the fourth or fifth left intercostal space, as close to the sternum as possible, with a fine needle.

8. That pericardial dilatation does not alter the relations of the pleuræ to the anterior thoracic wall.

9. In the presence of pyopericarditis the overlying pleural space is usually obliterated by extension of the inflammation.

10. That incision under local anæsthesia is the preferable operation in the majority of cases.

11. That Roberts' chondroplastic flap is preferable to an ordinary excision of costal cartilages when incision fails to give adequate drainage.